REPORT OF A CASE OF TABES WITH HEPATIC CRISES; AUTOPSY.*

By WILLIAM C. KRAUSS, M.D.,

BUFFALO, N. Y.

In a personal experience with twenty-two cases of tabes dorsalis, I have met with all the major symptoms and nearly all the minor ones of this disease, as depicted in the treatises on nervous diseases and in the special monographs on this subject. In only one case have I encountered phenomena not elsewhere observed by me, and as far as I am able to learn, not heretofore described. It may be presumptuous to call the attacks noted,—crises, or to regard them as part and parcel of the tabetic syndrome, but they resembled closely other commonly observed crises in character and sequence, that I unhesitatingly connect them with the disease, and will characterize them as hepatic crises.

The following peculiar and distressing attacks have been differentiated by various observers, and characterized by Charcot as crises tabetiques; they have been called respectively, according to the organ or part affected, gastric, laryngeal, pharyngeal, bronchial, cardiac, nephralgic, intestinal, vesical, rectal, clitoric or urethral crises. These all agree in so far that they are periodic, paroxysmal, limited to the one organ, are accompanied by pain and motor activity, and call forth the special function of the organ involved. Thus, for instance, the rectal crises are attacks of intense pain in the rectum with tenesmus, and accompanied by a sensation as if a foreign body were to be expelled. Hence in hepatic crises one would expect to find periodic paroxysms of pain over the hepatic area, with jaundice and clay-colored stools not referable to any diseased condition of the liver itself. Such, in truth, were the symptoms observed in the following interesting case:

Mrs. G.; age, 44 years; height, 5 feet 3 inches; weight, 138 pounds; complexion dark.

Family history was negative.

Early history: From childhood to commencement of present trouble she had always enjoyed good health. She gave no history of syphilis, either hereditary or acquired. She menstruated when 14 years old, and married when 23. About eight years before her death she began to have shooting pains in her legs, occurring at intervals and disappearing. Gradually difficulty in locomotion developed, so that she was obliged to walk with a cane, then with one crutch, then two crutches, and on this account she sought admission into the St. Francis' Hospital for the Chronic Sick and Aged in 1890. About this time she began to have excruciating pains over the right side of the chest, close to the lower border of the ribs, accompanied by a pronounced jaundice. These spells occurred every four to five weeks, obliging her to keep her bed for two to three days at a time, and then would pass off slowly, leaving her weak and exhausted for several days. The pain was diffuse, neuralgic in character, uninterrupted, deep-seated and very intense, necessitating the administration of large doses of morphia. The jaundice would remain for from four to six days, then would totally disappear. These spells were not accompanied by any nausea, vomiting or gastric disturbance of any kind. She usually partook of fluid food at these times without experiencing any distress; her bowels were regular, stools were clay-colored during these crises; at other times they were normal, and the urine showed traces of bile pigment, but no sugar or albumin.

---

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.

An examination of her condition in 1890 revealed the following symptoms: Mind was active and acute, memory was good, and she slept well. Her head was not painful to percussion, and she gave no history of headaches.

Eyes: Pupils were contracted; Argyll-Robertson pupil was present. No history of ptosis, double vision, or dimness of vision could be obtained and the ophthalmoscopic examination showed no atrophy of the optic nerve.

No disturbances of the senses of smell, taste or hearing were discoverable.

Strength of the hands was well preserved. No marked inco-ordination of the movements of the hands or arms was seen, and she was able to do some sewing and to make buttonholes for coatmakers. The legs, on the contrary, showed an extreme degree of inco-ordination, the gait was markedly ataxic, Romberg's symptom was present and the tendon reflexes were abolished. No disturbances of sensation existed, and the vesical and rectal reflexes were also undisturbed.

Palpation of the abdomen revealed no hepatic enlargement, no tenderness over the hepatic area, or over the abdomen. This was in marked contrast to the condition present during one of the crises. The hepatic area was then so sensitive that she could not bear the weight of her clothing, and she immediately went to bed when the attack was about to begin.

Examination of the feces showed no presence of bile, and no gall stones. The urine was highly colored, and gave the bile reactions.

During these attacks, the skin, mucous membranes, conjunctivæ, and even the scalp were all highly jaundiced, and she complained of the bitter taste in her food.

The attack lasted two or three days, and the intense pain was partially relieved by large doses of morphia hypodermically administered. Her condition following one of these attacks was one of great debility and prostration for fully a week. She then slowly recovered and was able to sew and take her daily suspensions.

The ataxia became so pronounced that she was unable to walk from her bed to the chair, and eventually had to be carried, until she became totally bedridden.

She gradually lost in strength; the hepatic disturbance became more and more intense, and in April, 1894, she died from inanition.

The autopsy was made the day after her death; special attention being directed to the brain, spinal cord and liver.

The brain was apparently unaffected; the dura and pia were moderately injected, were not adherent, and the Pacchionian bodies were not enlarged.

The spinal cord was not abnormal to the eye, and after the removal of the dura, the cord was placed in Müller's fluid for microscopical examination.

The heart and lungs, likewise the abdominal viscera, were normal in size, appearance and consistency. The liver was not enlarged, and weighed 3¼ pounds. Its capsule was not adherent, and on section was firm and uniform throughout. The hepatic vessels were moderately engorged with blood. The cut surface showed slightly the mottled appearance characteristic of nutmeg liver, and the hepatic and portal vessels contained a large quantity of blood.

The gall bladder was filled with bile, contained no gall stones, and the ductus choledochus and accessory ducts were passable with a sound.

The liver was cut into small cubes about one inch thick, so that no new growth of any kind would be overlooked, and many of these cubes were placed in alcohol and Müller's fluid for further examination.

After the usual hardening and imbedding methods had been employed, the liver and spinal cord were cut and stained with the carmine, hematoxylin, and by Weigert's method, and mounted in balsam.

The spinal cord sections showed sclerosis of the posterior tracts throughout the entire length of the cord. The peripheral nerves were not examined.

The liver appeared to be in the early stages of chronic congestion, otherwise known as nutmeg liver. There was, however, little atrophy of the cells about the central vein, and but very little pigment scattered throughout the cells. The vessels were not particularly engorged, nor had any hypertrophy taken place in their walls.

As a result of the microscopical examination, we find the characteristic lesions in the posterior tracts of the cord, and a nutmeg liver in its early or imperfect state.

The latter was without any cardiac or pulmonary disease, or any new growth producing pressure on the inferior vena cava or obstructing the return of venous blood to the thoracic organs. Neither were any of the sequelæ or concomitants of chronic venous engorgement present; such as ascites, hemorrhoids, varices, intestinal catarrh or diarrhea.

No condition was present, therefore, which would explain the mechanism of this congestion, which was purely a mechanical one. On the other hand, no condition was present in the liver, to explain the symptoms presented by this patient. No neoplasm of any kind, no hepatitis, and no gall stones were found. A chronic congested liver is not accompanied by any pain whatever, does not produce a very high degree of jaundice, does not cause paroxysmal symptoms, and can often be diagnosed by its enlargement.

It would seem that some disturbance to the nerve supply of the liver and gall bladder was present in this case, possibly producing constriction of the bile ducts and a consequent damming back of the bile secretion.

The occurrence of crises in tabes dorsalis is rather rare, as in the twenty-two cases observed by me, only one other case presented these symptoms, and they were referable to the stomach; were gastric crises. Riley[1] in his study of sixty-one cases of tabes found gastric crises in eleven cases and laryngeal crises in six cases. No other crises were observed. Olivet,[2] in studying the cases of tabes at the Göttingen clinic from 1877 to 1889, forty-three cases in all, found only four patients suffering from gastric crises. In the four hundred cases studied by Leimbach[3] in Professor Erb's clinic, only six per cent. were found suffering with tabetic crises.

As a rule the crises affect only one organ, but Grabower[4] reported a case to the Berlin Neurological Society in 1896, in which laryngeal, pharyngeal and gastric crises were present in the same patient.

---

[1] Journal of Nervous and Mental Disease, Sept., 1898.

[2] Inaugural Dissertation, Univ. Göttingen, 1891.

[3] Zeitschrift f. Nervenheilkunde, 1895, VII.

[4] Neurologisches Centralblatt, Feb. 1. 1896.

DISCUSSION.

Dr. William Osler inquired whether any fever accompanied the patient's attacks of liver trouble, and how long before her death they had ceased. The pain and jaundice accompanying the attacks would lead one to suspect the presence of gall stones. Even though no gall stones were found at the necropsy that did not exclude the possibility that they were present during life and gave rise to the hepatic symptoms.

Dr. Krauss, in answer to Dr. Osler, said the patient had had no fever whatever during her hepatic crises, and she had had these attacks until the time of her death. In fact, she was in a jaundiced condition when she died. The fæces were examined almost every day, but no traces of gall stones were ever found.

MINUTE ON THE DEATH OF DR. E. C. SEGUIN.

The committee appointed by the American Neurological Association to take action regarding the death of the late Dr. Edward Constant Seguin report the following minute:

The association has learned with deep sorrow of the death of Dr. Seguin. He was one of the charter members of the association; for many years he acted most efficiently as its secretary and treasurer; and he was its president at the fifteenth annual meeting held in 1889.

Some of his most valuable contributions to neurological medicine were in the form of papers read before this association, and he frequently took part in the debates of the association, his remarks always being well considered and of permanent value. The older members of the association will recall the continuous and close interest which he took in its proceedings. He was courteous in his bearing, but was courageous in the assertion of his opinions and in the maintenance of his positions. He was always appreciative of the merits of his colleagues.

As a teacher and writer his work is of the very highest order. As a neurological practitioner he has probably never been excelled. No physician ever gave closer or more painstaking attention to those by whom he was consulted.

In common with his father he took great interest in the study both of idiocy and of medical thermometry, adding much of great value to our knowledge of these subjects. He was largely instrumental in introducing to the profession in this country the use both of general and surface thermometry.

Dr. Seguin's contributions to neurology and to general medicine have given him a prominent position in medical literature. He and one or two others may be regarded as the pioneers of American neurology. His contributions to the therapeutics of nervous diseases are especially valuable. His work entitled "Opera Minora" contains a number of papers of great scientific and practical value. His pathological investigations into the nervous system were among the most important in the history of American neurology in the first ten years of the existence of this association.

J. J. Putnam,
L. C. Gray,
C. K. Mills.

THE FORMATION AND EXCRETION OF THE METAPLASM GRANULES OF THE NEURON.*

By Ira Van Gieson, M.D.

Dr. Van Gieson described the constituents of the ganglion cells and referred to the reticulum, which transmits the impulses and subserves the phenomena of retraction and extension of the neuron. The speaker said that when the food supply of the ganglion cell is diminished—as it is in alcoholism, in over-fatigue, etc.—we get an indication of it in an excretion of particles which escape from the cell, pass out of the lymph space and cluster about the blood vessels. These metaplastic elements are the results of over-fatigue—of a diminished food supply to the nerve cells. Dr. Van Gieson said that the chemic processes which accompanied this destructive metaplasm of ganglion cells must still be worked out. He had come to realize the fact that these nerve changes were not altogether of toxic origin, but that we must also take into consideration the quantitative and qualitative food supply of the cells.

Dr. Van Gieson thought that in amaurotic family idiocy there was an indication of a diminished food supply to the nerve cells. He agreed with Dr. Sachs that it was bad to make a distinction between arrested development and pathological processes. If we find a degenerative process in an adult, we call it by its proper name; in a child, we call it arrested development. One is the result of the other; degeneration is the result of arrested development. He suggested the name of chronic parenchymatous degeneration in infancy.

---

*Read at the twenty-fourth annual meeting of the American Neurological Association, May, 1898.